UNIVERSITY OF CALIFORNIA, IRVINE

BERKELEY • DAVIS • IRVINE • LOS ANGELES • RIVERSIDE • SAN DIEGO • SAN FRANCISCO

SANTA BARBARA • SANTA CRUZ

DEPARTMENT OF CHEMISTRY

IRVINE, CALIFORNIA 92717-2025

July 18, 1997

Richard Becker, Ph.D.
Director
Office of Environmental
Health Hazard Assessment
301 Capitol Mall, Second Floor
Sacramento, California 95814

Dear Dr. Becker:

On behalf of the Scientific Review Panel (SRP/Panel) I am pleased to transmit to you our Findings as a result of our review of the Office of Environmental Health Hazard Assessment (OEHHA) final report "Health Effects of Exposure to Environmental Tobacco Smoke" (ETS).

As you will see in a review of the SRP meeting transcript, the Panel is very impressed with the quality of the report and view it as the most current and definitive statement of the science applicable to ETS. As we noted OEHHA staff scientists are to be highly commended for this successful completion.

We are also pleased that the Air Resources Board (ARB) is considering holding an "informational hearing" on the report. As you will see in the enclosed Findings, the Panel views ETS as a toxic air contaminant, and it has a major impact on public health.

If the Panel may be of further help as this health risk is addressed in California, we would be pleased to do so.

We trust our Findings and this transmittal letter will be made a part of the final report

Sincerely,

James N. Pitts, Jr. Ph.D.
Chairman
Scientific Review Panel

Enclosure

cc: John D. Dunlap, Chairman, ARB
Scientific Review Panel Members
Bill Lockett, ARB

Findings of the Scientific Review Panel on
HEALTH EFFECTS OF EXPOSURE TO ENVIRONMENTAL TOBACCO SMOKE
as Adopted at the Panel's June 19, 1997 Meeting

The Scientific Review Panel (SRP/Panel) has reviewed the report "Health Effects of Exposure to Environmental Tobacco Smoke" prepared by the Office of Environmental Health Hazard Assessment (OEHHA). The Panel members also reviewed the public comments received on this report. Based on this review, the SRP makes the following findings:

1. Environmental Tobacco Smoke (ETS) is an important source of exposure to toxic air contaminants. Thus, despite an increasing number of restrictions on smoking and increased awareness of health impacts, exposures continue to be a major public health concern.

2. A causal association exists between ETS exposure from spousal smoking and coronary heart disease (CHD) mortality in nonsmokers. Risks associated with ETS exposure were almost always strengthened by adjustment for other cofactors. For nonsmokers exposed to spousal ETS compared to nonsmokers not exposed, the risk of CHD mortality is increased by a factor of 1.3. The association between CHD and risk is stronger for mortality than for non-fatal outcomes, including angina. Heart disease is the primary fatal endpoint from ETS exposure.

3. ETS is a complex mixture of chemicals generated during the burning and smoking of tobacco products. Chemicals present in ETS include irritants and systemic toxicants, mutagens and carcinogens, and reproductive and developmental toxicants. To date, over 50 compounds in tobacco smoke have been identified as carcinogens and six as developmental or reproductive toxicants under California's Proposition 65 (California Health and Safety Code 25249.5 *et seq.*) and twelve have been identified as a toxic air contaminant under AB 1807.

4. The 1986 *Report of the Surgeon General,* the 1986 National Research Council report *Environmental Tobacco Smoke: Measuring Exposures and Assessing Health Effects,* and the 1992 U.S. EPA report *Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders* have established that ETS exposure causes lung cancer. Results from recent epidemiological studies are compatible with the causal association already established.

5. Available data suggest that the prevalence of ETS exposure in California is lower than elsewhere in the U.S. Nevertheless, among adults in California, the workplace, home and other indoor locations all contribute significantly to ETS exposure. For children the most important single location is the home.

6. ETS exposure adversely affects fetal growth, with elevated risks of low birth weight or "small for gestational age" observed in numerous epidemiological studies. The primary effect observed, reduction in mean birth weight, is small in magnitude. If the distribution of birth weight is shifted lower with ETS exposure, as it appears to be with active smoking, infants who are already compromised may be pushed into even higher risk categories. Low birth weight is associated with many well-recognized problems for infants and is strongly associated with perinatal mortality.

7. Numerous studies have demonstrated an increased risk of sudden infant death syndrome, or "SIDS," in infants of mothers who smoke. Until recently it has not been possible to separate the effects of postnatal ETS exposure from those of prenatal exposure to maternal active smoking. Recent epidemiological studies now have demonstrated that postnatal ETS exposure is an independent cause of SIDS.

8. ETS exposure produces a variety of acute effects involving the upper and lower respiratory tract. In children, ETS exposure can exacerbate asthma, and increases the risk of lower respiratory tract illness, and acute and chronic middle ear infection. Eye and nasal irritation are the most commonly reported symptoms among adult nonsmokers exposed to ETS. Odor annoyance has been demonstrated in several studies.

9. Regarding chronic health effects, there is compelling evidence that ETS is a risk factor for induction of new cases of asthma as well as for increasing the severity of disease among children with established asthma. In addition, chronic respiratory symptoms in children, such as cough, phlegm, and wheezing, are associated with parental smoking. While the results from all studies are not wholly consistent, there is evidence that childhood exposure to ETS affects lung growth and development, as measured by small, but statistically significant decrements in pulmonary function tests; associated reductions may persist into adulthood.

10. The effect of chronic ETS exposure on pulmonary function in otherwise healthy adults is likely to be small. However, in combination with other insults (*e.g.*, prior smoking history, exposure to occupational irritants or ambient air pollutants), ETS exposure could contribute to chronic respiratory impairment in adults. In addition, regular ETS exposure in adults has been reported to increase the risk of occurrence of a variety of lower respiratory symptoms (e.g. bronchitis and wheezing apart from colds).

11. Children are especially sensitive to the respiratory effects of ETS exposure. Children with cystic fibrosis are likely to be more sensitive than healthy individuals. Several studies of patients with cystic fibrosis, a disease characterized by recurrent and chronic pulmonary infections, suggest that ETS can exacerbate the condition. Several studies have shown an increased risk of atopy (a predisposition to develop IgE antibodies against common allergens, which can then be manifested as a variety of allergic conditions) in children of smoking mothers, though the evidence regarding this issue is mixed.

12. Of the studies examining the effect of ETS exposure on nasal sinus cancers, all three show consistent associations, presenting strong evidence that ETS exposure increases the risk of nasal sinus cancers in nonsmoking adults. Further study is needed to characterize the magnitude of the risk of nasal sinus cancer from ETS exposure.

13. The epidemiological and biochemical evidence suggest that exposure to ETS may increase the risk of cervical cancer. Positive associations were observed in two of three case-control studies and a statistically nonsignificant positive association was observed in the only cohort study conducted. Findings of DNA adducts in the cervical epithelium as well as nicotine and cotinine in the cervical mucus of ETS-exposed nonsmokers provides biological plausibility.

14. Studies on ETS exposure and breast cancer suggest an association, but the associations were present only in select groups, or there is either no association between active smoking and the risk of breast cancer or the association for active smoking is weaker than for passive smoking. However, there is no indication of increasing risk with increasing intensity of ETS exposure. Still, results from a recent study suggest that tobacco smoke may influence the risk of breast cancer in certain susceptible groups of women, and this requires further investigation.

15. In summary, ETS exposure is causally associated with a number of fatal and non-fatal health effects. Heart disease mortality, sudden infant death syndrome, and lung and nasal sinus cancer have been causally linked to ETS exposure. Serious impacts of ETS on the young include childhood asthma induction and exacerbation, bronchitis and pneumonia, middle ear infection, chronic respiratory symptoms, and low birth weight. In adults acute and chronic heart disease morbidity is causally associated with ETS exposure. ETS also causes eye and nasal irritation and odor annoyance.

16. Effects for which evidence is suggestive of an association, but further research is needed for confirmation, include: spontaneous abortion, adverse neuropsychological development, cervical cancer, exacerbation of cystic fibrosis, and decreased pulmonary function.

17. It is not possible to judge on the basis of the current evidence the impact of ETS on a number of endpoints, including congenital malformations, changes in female fertility and fecundability, male reproductive effects, rare childhood cancers and cancers of the bladder, breast, stomach, brain, hematopoietic system, and lymphatic system.

18. Many Californians are exposed to ETS, and the number of people adversely affected is correspondingly large. Each year ETS contributes to asthma exacerbation in 48,000 to 120,000 children, 960 to 3120 new cases of asthma in children, 78,600 to 188,700 physicians office visits due to middle ear infections in children, 18,000 to 36,000 cases and 900 to 1800 hospitalizations from bronchitis or pneumonia in toddlers and infants, and 1,200 to 2,200 cases of low birth weight. Annual mortality estimates associated with ETS

exposure in California are: Approximately 120 deaths from SIDS, 16-25 deaths in toddlers and infants from bronchitis and pneumonia, approximately 360 deaths from lung cancer, and 4,200 - 7,440 deaths from ischemic heart disease. Thus, ETS has a major public health impact.

After careful review of the February 1997 draft of the OEHHA report, "Health Effects of Exposure to Environmental Tobacco Smoke," we find the draft, with the changes specified by OEHHA in our June 19, 1997 meeting, as representing a complete and balanced assessment of current scientific understanding. Based on the available evidence we conclude ETS is a toxic air contaminant.

I certify that the above is a true and correct copy of the findings adopted by the Scientific Review Panel on June 19, 1997

James N. Pitts, Jr., Ph.D.
Chairman, Scientific Review Panel